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Original Article

Evaluation of Salivary Inflammatory Biomarkers in Periodontitis Before and After Non-Surgical Therapy

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ABSTRACT

Background and Objective: Periodontal disease is defined as a chronic inflammatory condition characterized by gradual breakdown of the supporting structures of the tooth, including connective tissue and alveolar bone. Salivary inflammatory biomarkers, including matrix metalloproteinase-9 (MMP-9), prostaglandin E_2 (PGE_2), and alkaline phosphatase (ALP), have key functions in the inflammatory process, collagen breakdown, and bone remodeling. Evaluating their levels in relation to periodontal health and after treatment may provide clinically relevant information regarding disease progression and therapeutic effects. This research aimed to investigate the impact of nonsurgical periodontal therapy (NSPT) on salivary levels of MMP-9, PGE_2, and ALP in patients with generalized moderate-to-severe periodontitis, and to determine their relationships with clinical/periodontal parameters.

Material and Methods: Sixty participants meeting the inclusion criteria were enrolled and assigned to two groups: a control group (n = 30) exhibiting a clinically healthy periodontium, and a study group (n = 30) clinically confirmed to have generalized moderate-to-severe periodontitis. Periodontal clinical parameters comprising Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Loss (CAL), along with salivary levels of MMP-9, PGE_2, and ALP, were recorded at baseline for both groups. The study group underwent scaling and root-surface debridement, followed by a six-week follow-up assessment to reevaluate post-treatment clinical changes. An enzyme-linked immunosorbent assay (ELISA) was used to assess salivary inflammatory biomarker levels.

Results: At baseline, periodontitis patients displayed substantially higher clinical parameters than controls ($p < 0.005$), and higher salivary inflammatory biomarker levels: MMP-9: 15.36 ± 10.50 pg/mL, PGE_2: 525.70 ± 261.65 pg/mL, ALP: 0.55 ± 0.13 pg/mL. A direct positive association was observed between salivary biomarkers and disease severity. Both periodontal parameters and salivary biomarker levels decreased notably: MMP-9: 6.28 ± 5.28 pg/mL, PGE_2: 271.17 ± 129.09 pg/mL, and ALP: 0.36 ± 0.10 pg/mL ($p < 0.005$). The association between salivary biomarkers and clinical parameters became weaker or even inverse after nonsurgical periodontal treatment ($p < 0.005$).

Conclusion: The simultaneous improvement in clinical parameters and decline in salivary MMP-9, PGE_2, and ALP levels underscores their value as non-invasive indicators for tracking periodontal disease progression and evaluating therapeutic responsiveness.

Keywords: ALP, MMP-9, Nonsurgical periodontal therapy, Periodontitis, PGE2, Salivary inflammatory biomarkers.

1 Introduction

Periodontal disease is defined as a chronic, multifactorial inflammatory condition affecting the structures supporting the teeth. It is marked by the gradual breakdown of the periodontal ligament and alveolar bone, which ultimately leads to tooth loss if left untreated [1]. The disease results from a complex interaction between the subgingival microbial biofilm and host immune–inflammatory responses, influenced by genetic, environmental, and lifestyle factors [2]. Conventional periodontal diagnostic methods, such as Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Level (CAL), are commonly used; however, they have limitations in assessing real-time disease activity or predicting future periodontal tissue breakdown [3]. Consequently, increasing attention has been directed toward identifying reliable non-invasive biomarkers capable of supplementing clinical assessment, evaluating disease activity, and monitoring responses to periodontal therapy.

Saliva has emerged as a promising diagnostic medium because it is easy to collect, cost-effective, and contains a diverse spectrum

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of biomolecules derived from the oral cavity as well as systemic circulation [4]. Several salivary biomarkers associated with periodontal disease have been proposed to reflect ongoing inflammation, tissue degradation, and bone turnover. Among these, matrix metalloproteinase-9 (MMP-9), prostaglandin E_2 (PGE_2), and alkaline phosphatase (ALP) have been identified as potential markers. MMP-9 is a proteolytic enzyme involved in the degradation of extracellular matrix components during inflammatory tissue destruction [5]. PGE_2, a lipid-derived inflammatory mediator, induces vasodilation, pain, and bone resorption, and plays a key pro-inflammatory role in periodontal disease [6]. ALP is an enzyme associated with bone metabolism and mineralization and may reflect both periodontal tissue destruction and reparative processes [7].

Nonsurgical periodontal therapy (NSPT), which primarily includes scaling and root-surface debridement, represents the cornerstone of periodontal treatment aimed at reducing microbial load and controlling inflammation [8]. However, the effects of periodontal therapy on salivary biomarker levels remain inconsistent in the literature and warrant further investigation [9]. Salivary biomarkers offer a non-invasive approach for detecting and monitoring periodontal disease activity; nevertheless, limited data are available regarding their post-treatment changes and their association with clinical periodontal outcomes. Therefore, this study aims to evaluate the diagnostic and monitoring potential of salivary MMP-9, PGE_2, and ALP by assessing their levels in patients with periodontitis before and six weeks after NSPT, comparing them with periodontally healthy controls, and investigating their associations with clinical periodontal parameters.

2 Materials and Methods

2.1 Study Design and Setting

This research was conducted as a prospective controlled clinical trial at the Department of Periodontics, College of Dentistry, Hawler Medical University, Erbil City, Iraq. Patients were recruited directly from this department during the study period from January 2025 to June 2025. The study protocol was approved by the Ethical Committee of the College of Dentistry, Hawler Medical University (Ethics approval number: HMUD/2425054; date of approval: 22/12/2024) prior to study initiation. Written informed consent was obtained from each enrolled participant before participation.

2.2 Sample Size Calculation

An a priori sample size calculation was performed using G*Power software (Heinrich Heine University, Düsseldorf, Germany) to determine the sample size needed to detect statistically significant variances in salivary biomarker levels between study groups as well as before and after non-surgical periodontal therapy. The calculation was based on comparison of mean values, presuming a medium effect size based on previous studies on salivary inflammatory biomarkers, a significance level (α) of 0.05, and a statistical power ($1-\beta$) of 80%. On the basis of this analysis, a total sample size of 60 participants was deemed adequate to guarantee sufficient statistical power and the accuracy of the results.

2.3 Study Population

A total of 60 medically healthy individuals (27 males and 33 females), aged 30–60 years, participated in this study. Comprehensive medical and dental histories were recorded for all participants. Inclusion criteria were willingness to participate and having ≥20 teeth.

Exclusion criteria included systemic diseases that could affect periodontal status, pregnancy or breastfeeding, smoking, periodontal treatment within the previous 3 months, or the use of medications that could affect periodontal health.

Participants were allocated into two groups based on periodontal status: Control group: Thirty participants with no clinical signs of periodontal disease served as the control group, with PPD ≤ 3 mm and no CAL[10]. Study group: Thirty participants clinically confirmed to have generalized moderate-to-severe periodontitis were assigned to the study group, characterized by CAL ranging from 3–4 mm to ≥5 mm in more than 30% of sites, and PPD ranging from 4–7 mm [1].

2.4 Collection of Salivary Samples

All participants provided unstimulated whole saliva samples with passive drooling method between 9:00 and 11:00 am at baseline; for the study group, saliva was also collected after 6 weeks, following the method described by Mohammed et al. (2022) [11]. Participants were instructed to refrain from eating, drinking (except water), and performing oral hygiene procedures for at least 90 minutes before sample collection.

Each participant was seated comfortably in an upright position and allowed saliva to accumulate in the floor of the mouth before expectorating into a sterile graduated container until 5 mL of unstimulated saliva was obtained. Immediately after collection, samples were placed in an ice box, transported to the laboratory, and centrifuged at 4000 × g for 3 minutes. The supernatants were transferred into sterile Eppendorf tubes and stored at −30 °C until analysis.

2.5 Assessment of Inflammatory and Remodeling Biomarkers

To assess the levels of MMP-9, PGE_2, and ALP, enzyme-linked immunosorbent assay (ELISA) kits (ELK Biotechnology, USA) were utilized according to the manufacturer's instructions. The specific kits used were MMP-9 (ELK10238; sensitivity: 0.19 ng/mL; range: 23.44–1500 ng/mL), PGE_2 (ELK10997; sensitivity: 10.8 pg/mL; range: 31.25–2000 pg/mL), and ALP (ELK1941; sensitivity: 0.067 ng/mL; range: 0.16–10 ng/mL), all of which maintained intra-assay coefficients of variation (CVs) <8% and inter-assay CVs <10%. For each assay, lyophilized

standards were used to generate standard curves, allowing for the determination of sample concentrations based on these calibration curves. Final absorbance was measured at 450 nm using a microplate reader.

2.6 Evaluation of Clinical Periodontal Parameters

All participants underwent a comprehensive full-mouth periodontal evaluation at baseline; the study group was re-evaluated after 6 weeks following scaling and root-surface debridement. Clinical periodontal examination included assessment of PI and GI according to the criteria described by Löe and Silness (1963) and Silness and Löe (1964), respectively [12, 13]. Plaque Index and GI were assessed at four sites per tooth (buccal/facial, mesial, distal, and lingual/palatal).

Probing pocket depth and CAL were recorded according to Newman and Carranza (2023) [14] using a Williams periodontal probe (Medesy, Italy). Measurements were taken at six sites per tooth (mid-buccal, mesio-buccal, disto-buccal, mesio-lingual, disto-lingual, and mid-lingual). All values were recorded to the nearest millimeter. Periodontitis was defined by CAL ranging from 3–4 mm to ≥5 mm in more than 30% of sites, and PPD ranging from 4–7 mm [1].

To reduce measurement bias, the clinician performing the periodontal examination and the laboratory professional performing the ELISA analysis were blinded to group allocation and periodontal status.

2.7 Non - Surgical Periodontal Treatment

At baseline, full-mouth scaling and root-surface debridement were performed for patients in the study group using an ultrasonic scaler (Woodpecker, China) and Gracey curettes (Medesy, Italy) to remove supragingival and subgingival calculus and bacterial deposits from root surfaces. Oral hygiene instructions were provided to all patients.

Particular attention was paid to achieving smooth, biologically compatible root surfaces, which were checked using the tip of a periodontal probe, while avoiding unnecessary removal of cementum. Professional prophylaxis was then performed using a rotary handpiece and rubber cup to remove stains, followed by polishing with pumice powder.

A case of moderate periodontitis treated with scaling and root-surface debridement at baseline and re-evaluated is shown in Figure 1. During the 6-week post-treatment period, no antibiotics, anti-inflammatory drugs, or chemical plaque-control products were prescribed.

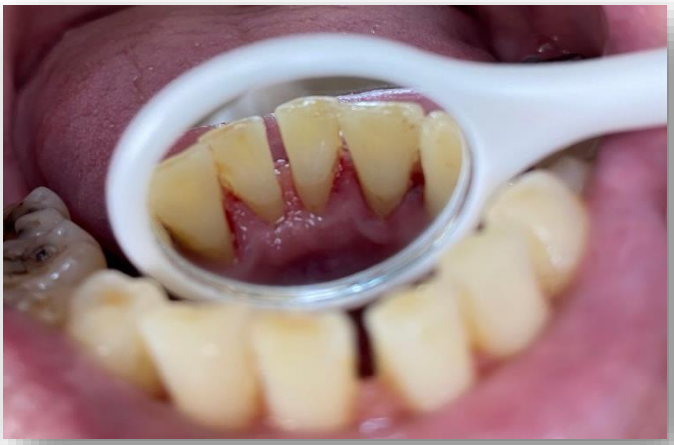

Figure 1: Clinical presentation of a case of moderate periodontitis before and after non-surgical periodontal therapy. (A) Baseline clinical photograph demonstrating extensive supragingival and subgingival calculus accumulation at the gingival margins, accompanied by edematous and erythematous gingival tissues prior to scaling and root debridement (SRD). (B) Clinical photograph obtained 6 weeks post-SRD, showing a significant resolution of gingival inflammation, decreased erythema, improved gingival contour, and the absence of visible calculus deposits.

2.8 Statistical Analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS), version 26. Data distribution was assessed using the Shapiro–Wilk test, and nonparametric tests were applied when appropriate. The chi-square (χ^2) test was used to compare categorical variables, and Fisher's exact test was used when expected cell counts were < 5. The unpaired t-test was used to examine intergroup differences in mean values. The Mann–Whitney U test was applied to compare mean ranks between two groups. The Wilcoxon signed-rank test was used to compare paired medians within the same sample. Spearman's rho (ρ) correlation coefficient was computed to evaluate the strength and direction of associations. A p-value ≤ 0.05 was considered statistically significant.

3 Results

3.1 Demographic Profile of the Study Population

Table 1 presents the demographic characteristics of the study participants by age and gender in both the control and study groups. A total of 60 medically healthy participants were recruited and divided into two equal groups: the control group (n = 30) and the study group (n = 30). Participants' ages ranged from 30 to 60 years.

The study population comprised 33 females (55%) and 27 males (45%). There were no statistically significant differences in gender distribution between the study and control groups (p = 0.194), indicating a balanced gender allocation between the two groups. The mean age of participants in the control group was 44.9 ± 9.2 years, whereas the mean age of participants in the study group was 47.2 ± 7.9 years. Data analysis showed no statistically significant difference in age distribution between the two groups (p = 0.299), indicating that the study and control groups were comparable in terms of age structure.

Table 1: Baseline Demographic Characteristics and Statistical Comparison of the Study and Control Groups.

Variable	Study Group No. (%)	Control Group No. (%)	Total No. (%)	*p*-value
Age range (years)				
30–39	4 (13.3)	9 (30.0)	13 (21.7)	
40–49	14 (46.7)	11 (36.7)	25 (41.7)	
50–60	12 (40.0)	10 (33.3)	22 (36.7)	
Mean (SD)	47.2 (7.9)	44.9 (9.2)		0.299**
Gender				0.194*
Males	11 (36.7)	16 (53.3)	27 (45.0)	
Females	19 (63.3)	14 (46.7)	33 (55.0)	
Total	30 (100.0)	30 (100.0)	60 (100.0)	

Data are presented as frequency (percentage) unless otherwise specified. SD: Standard Deviation. * p-value calculated using the Chi-square test for categorical variables (Gender). ** p-value calculated using the Independent. t-test for continuous variables (Mean Age). $p < 0.05$ is considered statistically significant.

3.2 Assessment of Clinical Periodontal Status in Study and Control Groups

Evaluation of clinical periodontal parameters between the control and study groups at baseline is presented in Table 2. The clinical findings showed that PI, GI, PPD, and CAL in the study group were 2.11 ± 0.61, 2.41 ± 0.57, 4.85 ± 0.64 mm, and 5.35 ± 0.71 mm, respectively. These values were significantly higher than those in the control group (0.85 ± 0.29, 0.98 ± 0.40, 0.00 ± 0.00 mm, and 0.88 ± 0.58 mm, respectively) ($p < 0.001$).

Table 2: Comparison of Baseline Clinical Periodontal Parameters Between Control and Study Groups.

Parameters	Control group (Baseline) M ±.SD	Study group (Baseline) M ±.SD	p-value
PI	0.85 ± 0.29	2.11 ± 0.61	< 0.001
GI	0.98 ± 0.40	2.41 ± 0.57	< 0.001
PPD (mm)	0.00 ± 0.00	4.85 ± 0.64	< 0.001
CAL (mm)	0.88 ± 0.58	5.35 ± 0.71	< 0.001

Data are expressed as Mean (M) ± Standard Deviation (SD). PI: Plaque Index; GI: Gingival Index; PPD: Probing Pocket Depth; CAL: Clinical Attachment Level. Statistical Analysis: p-values were determined using the Mann-Whitney U test for between-group comparisons at baseline. Significant at $p < 0.05$.

3.3 Evaluation of Salivary Inflammatory Biomarkers

Salivary inflammatory biomarker levels of MMP-9, PGE_2, and ALP were markedly elevated in the study group at baseline (15.36 ± 10.50 pg/mL, 525.70 ± 261.65 pg/mL, and 0.55 ± 0.13, respectively) compared with the control group (5.88 ± 3.37, 253.97 ± 106.65, and 0.26 ± 0.05, respectively) ($p < 0.001$), as shown in Table 3.

Table 3: Comparison of Baseline Salivary Inflammatory Biomarkers Between Control and Study Groups.

Biomarkers	Control group (Baseline)	Study group (Baseline)	p-value

	M ± SD	M ± SD	
MMP-9 (pg/mL)	5.88 ± 3.37	15.36 ± 10.50	< 0.001
PGE_2 (pg/mL)	253.97 ± 106.65	525.70 ± 261.65	< 0.001
ALP (pg/mL)	0.26 ± 0.05	0.55 ± 0.13	< 0.001

Data are presented as Mean (M) ± Standard Deviation (SD). MMP-9: Matrix Metalloproteinase-9; PGE_2: Prostaglandin E_2; ALP: Alkaline Phosphatase. Statistical Analysis: p-values were determined using the Mann-Whitney U test for between-group comparisons at baseline. Significant at $p < 0.05$.

3.4 Comparative Analysis of Salivary Inflammatory Mediators and Clinical Periodontal Status

Evaluation of clinical periodontal parameters and salivary biomarker levels in the study group at baseline and after NSPT is presented in Table 4. The clinical and biochemical findings showed significant improvements in the study group after 6 weeks of therapy compared with baseline values. Before treatment, PI, GI, PPD, and CAL were 2.11 ± 0.61, 2.41 ± 0.57, 4.85 ± 0.64 mm, and 5.35 ± 0.71 mm, respectively. After therapy, PI, GI, PPD, and CAL decreased to 1.03 ± 0.37, 1.13 ± 0.29, 2.12 ± 0.78 mm, and 2.98 ± 0.42 mm, respectively ($p < 0.001$). In line with these clinical improvements, salivary MMP-9, PGE_2, and ALP levels were markedly elevated at baseline (15.36 ± 10.50 pg/mL, 525.70 ± 261.65 pg/mL, and 0.55 ± 0.13, respectively). After treatment, MMP-9 decreased to 6.28 ± 5.28 pg/mL, PGE_2 decreased to 271.17 ± 129.09 pg/mL, and ALP decreased to 0.36 ± 0.10 ($p < 0.001$).

Table 4: Longitudinal Comparison of Clinical Parameters and Salivary Biomarkers in the Study Group Before and After Non-Surgical Therapy.

Parameter	Study group (Baseline) M ±.SD	Study group (After 6 weeks) M ±.SD	p-value
PI	2.11 ± 0.61	1.03 ± 0.37	< 0.001
GI	2.41 ± 0.57	1.13 ± 0.29	< 0.001
PPD (mm)	4.85 ± 0.64	2.12 ± 0.78	< 0.001
CAL (mm)	5.35 ± 0.71	2.98 ± 0.42	< 0.001
MMP-9 (pg/mL)	15.36 ± 10.50	6.28 ± 5.28	< 0.001
PGE_2 (pg/mL)	525.70 ± 261.65	271.17 ± 129.09	< 0.001
ALP (pg/mL)	0.55 ± 0.13	0.36 ± 0.10	< 0.001

Data are presented as Mean (M) ± Standard Deviation (SD). PI: Plaque Index; GI: Gingival Index; PPD: Probing Pocket Depth; CAL: Clinical Attachment Level; MMP-9: Matrix Metalloproteinase-9; PGE_2: Prostaglandin E_2; ALP: Alkaline Phosphatase. Statistical Analysis: p-values were determined using the Wilcoxon signed-rank test for intra-group longitudinal comparison. Significant at $p < 0.05$.

3.5 Association of Periodontal Status with Salivary Biochemical Profiles at Baseline and Post-Therapy

At baseline, salivary MMP-9, PGE_2, and ALP showed strong positive associations with all periodontal clinical parameters, including PI, GI, PPD, and CAL ($p < 0.001$). After therapy, these relationships shifted to weak positive or inverse patterns. For example, MMP-9 showed a significant positive association with GI; in contrast, it showed weak, non-significant positive associations with PI and CAL ($r = 0.140$ and $r = 0.035$, respectively) and a negative association with PPD ($r = -0.025$). Similarly, PGE_2 showed weak, non-significant positive associations with PI, GI, and PPD ($r = 0.107$, $r = 0.103$, and $r = 0.125$, respectively) and a negative association with CAL ($r = -0.256$). Alkaline phosphatase showed negative associations with PI, PPD, and CAL ($r = -0.139$, $r = -0.211$, and $r = -0.074$, respectively) and a non-significant weak positive association with GI ($r = 0.089$; $p = 0.05$), as presented in Table 5.

Table 5: Correlation Matrix Between Clinical Periodontal Parameters and Salivary Biomarkers Before and After Therapy.

	MMP-9		PGE_2		ALP	
Clinical Parameter	**(r)**	**P value**	**(r)**	**P value**	**(r)**	**P value**
Before treatment						
PI	0.548	< 0.001	0.687	< 0.001*	0.740	< 0.001*
GI	0.527	< 0.001	0.718	< 0.001*	0.679	< 0.001*
PPD	0.696	< 0.001	0.737	< 0.001*	0.784	< 0.001*
CAL	0.618	< 0.001	0.645	< 0.001*	0.754	< 0.001*

After treatment						
PI	0.140	0.460	0.107	0.572	–0.139,	0.462
GI	0.508	0.004*	0.103	0.588	0.089,	0.461
PPD	–0.025	0.894	0.125	0.510	–0.211	0.264
CAL	0.035	0.854	–0.256	0.171	–0.074	0.698

r = Spearman's rho correlation coefficient. * Correlation is statistically significant ($p < 0.05$). PI: Plaque Index; GI: Gingival Index; PPD: Probing Pocket Depth; CAL: Clinical Attachment Level; MMP-9: Matrix Metalloproteinase-9; PGE_2: Prostaglandin E_2; ALP: Alkaline Phosphatase.

4 Discussion

This study evaluated the impact of NSPT on clinical periodontal parameters and salivary inflammatory biomarkers and investigated the associations between these variables in patients with generalized moderate-to-severe periodontitis. In the present study, periodontal treatment consisted of nonsurgical procedures. Patients in the study group received scaling and root-surface debridement combined with comprehensive oral hygiene instructions. This approach remains the cornerstone of periodontal therapy because it mechanically removes plaque and calculus, reduces bacterial burden, and decreases inflammatory mediators within gingival tissues. Consequently, gingival inflammation diminishes, probing pocket depth decreases, and clinical attachment levels may be partially restored through tissue repair and reattachment [15]. In line with this mechanism, the current study demonstrated significant improvement in clinical parameters, PI, GI, PPD, and CAL, after 6 weeks of treatment in the study group. These findings are consistent with previous studies reporting clinical improvement following 6 weeks of NSPT [16,17]. However, Kim (2022) reported no significant reduction in PPD after 6 weeks of scaling and root-surface debridement [18].

The present results also showed significantly higher salivary MMP-9 levels in the study group compared with the control group at baseline. A significant reduction in salivary MMP-9 was observed after 6 weeks of therapy, which may reflect resolution of inflammation and reduced recruitment and activity of polymorphonuclear neutrophils, the main cellular source of MMP-9 [5,19]. These findings agree with previous reports demonstrating elevated salivary MMP-9 levels in periodontitis patients compared with healthy controls at baseline, followed by a significant decline after NSPT [18, 11, 20, 21]. In contrast, Gonçalves et al. (2009) observed no significant difference in salivary MMP-9 levels between chronic periodontitis patients and controls at baseline [22]. Such discrepancies may be related to methodological differences in biomarker detection, sample size, disease extent/severity, and the clinical criteria used to define periodontal status.

Regarding PGE_2, the current study detected significantly elevated salivary PGE_2 levels in the study group compared with the control group at baseline, with a significant reduction after treatment. These findings suggest that scaling and root-surface debridement may attenuate the local inflammatory response by reducing activation of prostaglandin-mediated pathways, which are closely linked to vasodilation and alveolar bone resorption, thereby reflecting an improved host response to therapy [23]. Consistent with these results, Gümü et al. (2017) documented significantly higher salivary PGE_2 levels in patients with generalized chronic periodontitis [24]. Comparable outcomes were also reported by Shende et al. (2021), who assessed salivary PGE_2 levels in generalized chronic periodontitis before and after 6 weeks of Phase I periodontal therapy [25].

In addition, the current study found that patients with periodontitis exhibited elevated salivary ALP levels at baseline, followed by a significant decrease after NSPT. These findings are consistent with previous research that measured salivary ALP before and after 4 weeks of NSPT in patients with gingivitis and chronic periodontitis and reported a significant post-treatment reduction [26]. Nevertheless, not all literature aligns with these results. Mohammad and Aziz (2018) reported elevated salivary ALP levels in chronic periodontitis patients compared with non-periodontitis participants at baseline, with a significant reduction at 2 and 4 weeks after NSPT; however, they observed a marked increase after 6 weeks, suggesting inadequate maintenance of oral hygiene and possible relapse with renewed plaque accumulation [27]. Moreover, Yaghini et al. (2020) reported a non-significant change in ALP levels after NSPT, indicating that ALP may be influenced by multiple host- and treatment-related factors [28]. Differences between the present findings and those of Yaghini et al. (2020) may be attributed to variations in sample size and laboratory methodology, as their study included 16 patients and used a colorimetric assay for salivary ALP measurement, which differs from the ELISA-based method used in the current study [28].

At baseline, clinical periodontal parameters and salivary inflammatory biomarkers showed highly significant associations, indicating that greater plaque accumulation and attachment loss were directly linked to increased inflammatory activity. Therefore, elevated salivary MMP-9, PGE_2, and ALP levels in periodontitis patients—together with their strong associations with clinical indicators of periodontal damage support the potential role of these biomarkers as indicators of ongoing inflammation and tissue destruction [29]. After therapy, PI and GI showed weak associations with salivary biomarkers, which may reflect residual low-grade gingival inflammation, whereas associations with PPD and CAL appeared attenuated, possibly due to slower structural healing.

Conclusion

Patients with periodontitis exhibited significantly higher clinical periodontal parameters and salivary inflammatory biomarker levels at baseline compared with healthy controls. Following scaling and root-surface debridement, both clinical periodontal parameters and salivary inflammatory biomarkers decreased significantly in the periodontitis group. These findings support the effectiveness of NSPT in improving periodontal status and suggest that salivary biomarkers may serve as adjunctive tools for monitoring periodontal disease progression and response to therapy.

Limitations of the Study

A limitation of the present study is the relatively short 6-week follow-up period after NSPT, which limits evaluation of the intervention's long-term effects, as this interval may precede the stabilization phase of periodontal healing. Therefore, longer follow-up periods are recommended to assess the sustained impact of periodontal therapy on the salivary biomarker profile.

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